Cortical laminar necrosis mimicking leptomeningeal recurrence in a child with medulloblastoma and epilepsy

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To the Editor,

We report a case of cortical laminar necrosis mimicking leptomeningeal recurrence in a patient with medulloblastoma. Cortical laminar necrosis (CLN) is caused by cerebral energy depletion resulting in necrosis of a particular cortical laminar, usually the third layer.[1, 2] In children, it can be due to hypoxic-ischaemic encephalopathy, infarction, status epilepticus, Moya-Moya disease and metabolic disorders.[3] On T1 weighted imaging, CLN manifests as hyperintense lesions of the cerebral cortex in the subacute and chronic setting. Other hyperintense T1 weighted lesions for the differential diagnosis include: haemoglobin degradation products, lipids, cholesterol, high protein substances, melanin and certain minerals.[4] Understanding the radiological manifestations of different non-neoplastic intracranial pathologies, along with serial imaging and attention to the clinical picture are important to make accurate diagnoses and exclude potential mimics.

A seven-year-old male was diagnosed with metastatic anaplastic medulloblastoma (Fig. 1a-c). Initially, he only underwent complete resection due to parents declining adjuvant treatment. Three months later, the metastatic disease in the spine progressed and parents accepted further treatment. He received craniospinal irradiation (35Gy plus 15Gy boost to posterior fossa and metastases) with concomitant carboplatin and vincristine followed by vincristine/cyclophosphamide (6 cycles), as per the CCG-99701 trial.[5]At the end of treatment he was in complete remission.

The patient's ensuing long-term complications included multiple endocrinopathies (central precocious puberty, hypogonadotropic hypogonadism, growth hormone deficiency, central hypothyroidism, cortisol insufficiency) and secondary epilepsy (complex partial seizures).

Nine years post-diagnosis, a routine surveillance MRI scan identified what was then interpreted as new abnormal leptomeningeal enhancement in the right medial parietal and right temporo-occipital region suspicious of disease recurrence (Fig. 1g-i). The child was clinically stable, with no reported symptoms and no new focal deficits on examination. However on further review of his history, fifteen months earlier he had been admitted to his local hospital with status epilepticus requiring intubation and ventilation. The MRI during the admission had shown new abnormal changes in right cerebral hemisphere (Fig. 1d-f). These findings were attributed to prolonged, repetitive seizure activity, rather than to relapsed medulloblastoma. With the benefit of hindsight, the MRI findings 9 years post-diagnosis were improving compared to the previous scan, now showing intrinsic cortical T1 hyperintensity in keeping with CLN (Fig. 1g-i). A repeat MRI three months later showed stable intracranial appearances with no new signal abnormalities to suggest disease relapse, reinforcing the diagnosis of CLN (Fig. 1j-k).

Seizure-induced reversible MRI abnormalities (SRMA) have been described, usually with resolution within a few months. In some cases, there is widespread cortical enhancement, which can be mistaken for leptomeningeal enhancement. CLN is a long-term sequela of SRMA and radiological changes can sometimes persist for several years. [4,6] CLN can occur following hypoxia, hypoglycaemia, ischaemic stroke, anorexia, status epilepticus or after treatment with immunosuppressants or chemotherapy.[7,8]

The characteristics and evolution of SRMA are not clearly understood, hence the difficulty in distinguishing epilepsy-associated lesions from tumours or other pathology. The history, clinical presentation and serial imaging can aid in determining the natural history and evolution of CLN in such cases. This case highlights the resemblance of

non-neoplastic entities, such as SRMA and CLN, to leptomeningeal enhancement in malignant brain tumours. A careful evaluation of all MRI sequences including the non-contrast T1 weighted images and knowledge of potential mimics is required to aid the diagnostic process.

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